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09/087,513	05/29/98	KANEKO Y	0010-0929-0X

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ART UNIT	**PAPER NUMBER**
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Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary	Application No. 09/087,513	Applicant(s) Kaneko et al.
	Examiner Wilson, Michael C.	Group Art Unit 1633

☐ Responsive to communication(s) filed on _____.

☐ This action is **FINAL.**

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire 3 month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

Disposition of Claims

☒ Claim(s) 1-20 is/are pending in the application.

Of the above, claim(s) _____ is/are withdrawn from consideration.

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) 1-20 is/are rejected.

☐ Claim(s) _____ is/are objected to.

☐ Claims _____ are subject to restriction or election requirement.

Application Papers

☒ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The proposed drawing correction, filed on _____ is ☐approved ☐disapproved.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).

☐ All ☐ Some* ☐ None of the CERTIFIED copies of the priority documents have been

☐ received.

☐ received in Application No. (Series Code/Serial Number) _____.

☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

*Certified copies not received: _____.

☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

☒ Notice of References Cited, PTO-892

☐ Information Disclosure Statement(s), PTO-1449, Paper No(s). _____

☐ Interview Summary, PTO-413

☒ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Notice of Informal Patent Application, PTO-152

--- SEE OFFICE ACTION ON THE FOLLOWING PAGES ---

DETAILED ACTION

The sequence listing filed 1-7-00, paper number 6 ½, has been entered and is correct.

Claims 1-20 are pending and under consideration in the instant application.

Specification

1. The specification refers to a vv-ΔV3 mutant with the Δ297-329 deletion in 15 incorporated by reference herein in its entirety (page 26, line 17). However, reference 15 is not cited such that the reference can be determined. Deletion of "15" is suggested. Replacement of "15" with a reference not previously disclosed may require a new matter rejection.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

2. Claims 1-20 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claimed invention is drawn to a method of inducing a cellular immune response against a conserved epitope of a viral glycoprotein by administering DNA encoding a viral glycoprotein containing a modified immunodominant epitope, a method of making a vaccine against a virus and a vaccine for inducing cellular immunity against a virus. The specification identifies the essential feature of the invention is a viral envelope glycoprotein with a modified immunodominant epitope. Genes for various viral envelope glycoproteins and modifications to epitopes of viral envelope glycoproteins such as in the V3 loop in HIV were known in the art.

The specification discusses deleting the V3 loop of HIV-IIIB (page 26) described as a Δ297-329 deletion, but does not teach the nucleotide or amino acid sequence of HIV-IIIB, what applicants consider 297-329, which reference is "15" (line 17), the structural elements that are deleted or the structural elements of the pSVIII used to make the modification of the V3 loop or any other epitope in the instant invention. The specification discloses the 1ΔV3, 7ΔV3 and 8ΔV3 mutants (e.g. Example 14, page 34, Fig. 1) but does not teach how to make such mutants, how the mutants differ from each other, how the mutants differ from the vv-ΔV3 mutant with the Δ297-329 deletion (page 26) or the structural elements of the mutants. The specification discloses the WTP-2, WTP-5 and WTP-8 (page 35, line 3; page 36, line 16; Fig. 1), but it is unclear how the envelope gene in these vectors differs from each other or from the V3 mutants or whether these vectors are considered "modified". The specification does not disclose using these vectors or any cells expressing envelope glycoproteins containing modified immunodominant epitopes to induce a cellular immune response in a patient. The specification does not teach

which "conserved" epitope the cellular immune response is directed toward or one epitope that is "conserved" or "neutralizing". The specification teaches viruses included in the instant invention are influenza, visna virus and lentiviruses such as HIV, EIAV, CAEV, SIV, FIV (page 22, line 15). The specification does not teach how to modify influenza, visna virus, EIAV, CAEV, SIV or FIV envelope genes, any other strain of HIV envelope genes or any other viral envelope glycoprotein. Therefore, the specification does not provide adequate written description regarding the structure or function of "modified immunodominant," "conserved" or "neutralizing" epitopes that are essential to induce cellular immunity against a conserved epitope of said glycoprotein or against a virus or to obtain a therapeutic or prophylactic effect.

3. Claims 1-20 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The disclosed purpose of the instant invention is to provide therapy for patients with a viral infection, specifically HIV (page 1, line 12). The specification does not provide any other use for the method of inducing a cellular immune response, the vaccine or method of making a vaccine as claimed. The purpose of the specification is to guide the artisan on the making and using of the claimed invention, and the artisan reads the claims in light of the teachings in the specification. Thus, the artisan reading the claimed invention in view of the specification would only determine the use of the method of inducing a cellular immune response, a method of making

a vaccine and a vaccine to be for treating viral infection. If the Applicants feels other uses for the method of inducing differentiation are disclosed in the specification, then the applicants should point to such uses by page and line number. Otherwise, the field of the present invention is inducing cellular immunity to providing viral infection. While the art teaches the steps required to induce a cellular immune response against a conserved epitope, the art does not teach inducing a cellular immune response such that viral infections are treated. Therefore, the following enablement rejection is based on the sole disclosed use of inducing a cellular immune response against a conserved epitope of a viral envelope glycoprotein to treat viral infection, does not conflict with the art rejection below and is considered proper.

Claims 1-13 are directed toward inducing cellular immunity against a virus by administering a nucleic acid encoding an envelope glycoprotein of the virus and induces cellular immunity to a conserved epitope of the glycoprotein. Claims 14-18 are directed toward making a vaccine comprising a nucleic acid encoding an envelope glycoprotein of a virus. Claims 19 and 20 are directed toward a vaccine comprising cells expressing an viral envelope glycoprotein.

At the time of filing, it was unpredictable whether the production and administration of a nucleic acid constructs for the purpose of gene therapy would correlate to a therapeutic or prophylactic effect. Ross (September 1996, Human Gene Therapy, Vol. 7, pages 1781-1790) states a major technical impediment to gene transfer is the lack of ideal gene delivery systems including vectors, promoters and modes of delivery (page 1782, column 2, first full paragraph). These technical parameters are required to obtain efficient delivery and sustained expression of

the gene (Verma Sept. 18, 1997, Nature, Vol. 389, page 239-242; see page 239, 3rd column, line 10). The difficulties in sustaining expression of a gene cause an unpredictability in obtaining a therapeutic or prophylactic effect in a patient (Ross, page 1789, column 1, first paragraph).

Furthermore, Haynes (1993, Science, Vol. 260, pages 1279-1286) teaches the classic approach to vaccine development which involves exposing cells of the immune system to the proper antigenic stimulus required to stimulating a beneficial immune response. The prior art presents few examples where a single antigenic stimulus, such as a small limited peptide or a whole protein is found to engender a therapeutic or protective immune response. The successful art-recognized immunogens used as vaccines are derived from whole killed or live attenuated pathogens, are comprised of complex antigenic mixtures or comprise inactivated toxins. Many of these successes were achieved with a certain degree of luck, influenced by some particular peculiarity or aspect of a given pathogenic agent.

Regarding HIV vaccines, Stricker (Medical Hypotheses, June 1997, Vol. 48, pages 527-9; see page 527, last paragraph through all of page 528) teaches that attempts to develop a vaccine against HIV have been unsuccessful. In fact, HIV, SIV and FIV infection have defied the creation of an effective vaccine or immunotherapeutic. Overall, a lack of understanding about protective immunity to HIV, the sequence variability of HIV and the rapid replication of HIV, as disclosed by Bangham contribute the ineffectiveness of vaccines against HIV (Nov. 29, 1997, Lancet, Vol. 350, pages 1617-1621; page 1617, top of column 1). It is not known what renders an antigen capable of stimulating beneficial or protective CTL responses to lentiviruses, so the art at the time

of filing did not teach immunodominant antigens that could be used to vaccinate against lentiviruses such as FIV, SIV or HIV. Thus, the state of the art at the time of filing was such that it was unpredictable what parameters were required to administer a nucleic acid encoding a viral envelope glycoprotein or a cell expressing such a protein to a patient and obtain a cellular immune response against a viral epitope of HIV envelope glycoprotein that results in a therapeutic or prophylactic effect.

The specification discloses the vv-ΔV3 mutant which is described as having a Δ297-329 deletion but does not teach the nucleotide or amino acid sequence of HIV-IIIB, what applicants consider 297-329, which reference is "15" (line 17), the structural elements that are deleted or the structural elements of the pSVIII used to make the mutants. The specification discloses the 1ΔV3, 7ΔV3 and 8ΔV3 mutants (e.g. Example 14, page 34, Fig. 1) but does not teach how to make such mutants, how the mutants differ from each other, how the mutants differ from the vv-ΔV3 mutant with the Δ297-329 deletion (page 26) or the structural elements of the mutants. The specification discloses the WTP-2, WTP-5 and WTP-8 (page 35, line 3; page 36, line 16; Fig. 1), but it is unclear how the envelope gene in these vectors differs from each other or from the V3 mutants or whether these vectors are considered "modified". Therefore, the specification does not provide adequate guidance how to make the disclosed vectors, how the vectors differ from each other or the structural elements of the vectors.

The specification provides CTL and antibody-dependent cell-mediated cytotoxicity data *in vitro* (page 35-38), but does not provide any examples of inducing cellular immunity *in vivo* or

provide adequate correlative evidence between *in vitro* data and *in vivo* results such that a therapeutic or prophylactic effect against a virus could be obtained. It was well known in the art that assays measuring cellular immunity *in vitro* produce variable results depending on the target cells used, the effector to target ratio used, and the incubation time (Lancki, 1992, Biotherapy, Vol. 5, pages 71-81; see page 72, column 1, line 1) Cytotoxicity assays combine peripheral blood lymphocytes and target cells that are artificially "loaded" with antigen. The amount of antigen required on the target cell surface to induce a CTL response depends upon the immunostimulatory epitope of the antigen, the type of immune response and the strength of the immune response desired. Moreover, cytotoxicity assays do not account for the complex interaction of the immune response and cytokine regulation that occurs *in vivo*. For example, Bachmann reviews the use of the cellular immune response both *in vivo* and *in vitro* in viral assay systems (1994, Current Op. Immunol. Vol. 6, pages 320-326). A comparison of sensitivities shows that radioactive cytolytic assays are more sensitive than *in vivo* assays, but that results of secondary *in vitro* stimulation need to be verified by *in vivo* assay. On page 323, Bachmann states one should be very cautious not to 'over-interpret' results obtained by a cytolytic assay where cells are stimulated *in vitro* because the results may be biologically irrelevant without *in vivo* confirmation. It is unpredictable whether a cytotoxic response obtained *in vivo* equivalent to the cytotoxicity obtained *in vitro* will have any biological relevance. One of skill in the art would not warrant cytotoxicity assays *in vitro* to be an indicator of efficacy *in vivo*.

The *in vitro* CTL and ADCC assays disclosed in the instant application require PBMC isolated from an HIV patient and autologous B-LCL or Jurkat cells transfected with the vectors of the invention as target cells which do not correlate to cells or nucleic acids used to treat viral infection *in vivo*. The specification does not teach the strain of HIV in the patients used to make the PBMC *in vitro*, the level of antigen expression on the surface of target cells *in vitro*, the level of expression required *in vivo*, or how the immune response obtained *in vitro* correlates to response expected *in vivo*. It is not clear that the ratios of target to effector ratio used *in vitro* correlates to the ratio of transfected cells to effector cells that would occur *in vivo*. It addition, applicants activated the PBMC with antibodies which is an artificial means used to increase the activity of the cytotoxic cells and does not correlate to conditions found in the HIV patients because patients PBMCs are not stimulated with anti-CD3 antibodies. In addition, the specification does not teach that the level of cellular immunity *in vitro* would have any therapeutic benefit in a patient. Given the state of the art regarding the lack of correlation between *in vitro* and *in vivo* cytotoxicity taken with the guidance provided in the specification, it would have required one of skill undue experimentation to determine the parameters required to obtain an cellular immune response *in vivo* that has a therapeutic or prophylactic effect.

Claims 1-3, 5-15, 18 and 19 encompass modifying any viral envelope protein gene; claims 4 and 16 are limited to lentiviruses and claims 5, 17 and 20 are limited to any strain of HIV. The specification teaches viruses included in the instant invention are influenza, visna virus and lentiviruses such as HIV, EIAV, CAEV, SIV, FIV (page 22, line 15). The state of the art at the

time of filing was such that the envelope region of HIV, specifically the V3 region, varied between HIV strains (page 1, line 15; page 2, line 13). However, the specification only teaches modifying the HIV-IIIB glycoprotein gene. The specification does not teach how to modify influenza, visna virus, EIAV, CAEV, SIV or FIV envelope genes, any other strain of HIV envelope genes or any other viral envelope glycoprotein. Lentiviruses do not correlate to influenza or visna viruses because they do not have the same genome or surface proteins. Therefore, it is unclear how to modify any viral glycoprotein "immunodominant epitope" such that cellular immunity against a "conserved epitope" can be obtained that is therapeutic or prophylactic.

Applicants propose that a cellular immune response directed toward "conserved" epitopes of viral glycoproteins induces cellular immunity to multiple strains of virus (page 3, line 14). It is unclear how "cellular immunity to a conserved epitope" correlates to the "modified immunodominant epitope" in claim 1 because the specification does not teach how the V3 epitope is modified, how an epitope is "neutralizing", what epitope is being recognized in the cytotoxicity experiments or that the epitope recognized is "conserved" between HIV strains or other viruses. The specification does not correlate the HIV-IIIB envelope gene to influenza, visna virus, EIAV, CAEV, SIV or FIV envelope genes or other HIV viral envelope genes such similar modifications could be made or that cellular immunity could be induced against the protein. In fact, applicants demonstrate different modifications of HIV-IIIB cause different effects (e.g. 1ΔV3, 7ΔV3 or 8ΔV3 mutants induce different immune responses, Fig. 1). Therefore, the specification does not

enable one of skill to determine how to modify an epitope of a viral envelope glycoprotein in the methods or products claimed such that a cellular immune response against a "conserved" epitope resulting a vaccine, a therapeutic or prophylactic effect against the virus could be obtained.

Claim 1 is not enabled because cellular immunity is not directed against viruses; it is directed toward cells infected with viruses wherein said cells present viral antigens on their surface. In addition, claim 1 does not require antigen presentation or recognition of a viral protein by the cellular immune system which appears to be essential to the invention. Therefore, the claims do not clearly delineate the fact that the APC functionally express the modified protein such that cytotoxic cells recognize the modified protein on the surface of the APC and become specific to the envelope glycoprotein.

Overall, the specification does not provide adequate guidance regarding the epitope to modify, the epitope recognized, how to modify the epitope, the route of delivery, dosage, promoter, vector or the levels of protein expression required *in vivo* to induce cellular immunity to a conserved epitope *in vivo* such that a therapeutic or prophylactic effect is obtained. The specification does not teach how to make or use DNA or cells that are expressing a modified glycoprotein combined with an adjuvant such that a therapeutic or prophylactic effect is obtained. The specification does not teach how to stimulate PBMC in a patient, increase CTL activity in a patient such that a therapeutic effect is obtained. The specification does not teach the parameters required to use APCs that are resistant to ADCC, prevent syncytia formation, do not undergo apoptosis or prevent apoptosis of CD4+ T cells in a patient to treat or prevent viral infection

(claims 9-12). Given the state of the art taken with the guidance provided in the specification, it would require one of skill undue experimentation to use any nucleic acids or vaccines as claimed to induce a cellular immune response *in vivo* such that a therapeutic or prophylactic effect is obtained.

Therefore, in view of the lack of guidance in the specification regarding how to make or use the vaccines claimed, the lack of correlation between *in vitro* cytotoxicity and *in vivo* cytotoxicity, the unpredictability in the art regarding how to obtain a vaccine against viral infections, specifically HIV, the examples provided and the breadth of the claims, the ordinary artisan at the time of the instant invention would not have known how to make and/or use the claimed invention with a reasonable expectation of success.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claims 1-20 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1-13 are indefinite because cellular immunity is not directed against viruses or epitopes (claims 1 and 8); it is directed toward cells infected with viruses wherein said cells present viral antigens on their surface. It is unclear how "the conserved epitope" correlates to the "modified immunodominant epitope". Is the immune response directed toward the "modified

immunodominant epitope" found in other cells or is the immune response directed toward a different epitope that is "conserved"? The claim as written does not require antigen presentation or recognition of a viral protein by the cellular immune system. Therefore, the claim does not clearly delineate the difference between the "conserved epitope" and the "modified immunodominant epitope", what cells express the modified protein, what immune cells recognize the modified protein, and what epitope the immune cells are recognizing on the APCs.

The term "conserved epitope" is indefinite (claims 1 and 8). The envelope glycoproteins of viruses have considerable variations. For example, the third V3 loop of HIV envelope glycoprotein that appears to be the epitope to modify in the instant invention varies widely between strains of HIV and may even mutate within a patient to escape the immune system of the patient (page 2, line 13; page 3, line 4). The V3 loop may be the epitope recognized by the cellular immune system in the instant invention. However, the V3 loop is not conserved. Therefore, the cellular immune response may not be directed toward a conserved epitope. Thus, the metes and bounds of which epitopes are recognized by the cellular immune system cannot be determined.

The term "modified" is indefinite (claims 1, 9-12, 14 and 19). The third V3 loop of HIV varies widely between strains of HIV and may even mutate within a patient to escape the immune system of the patient (page 2, line 13; page 3, line 4). It is unclear whether "modified" epitopes encompass naturally occurring mutations or modifications of epitopes, man-made mutations in the

epitope or some other man-made "modification" to the epitope. Thus, the metes and bounds of what epitopes applicants consider "modified" cannot be determined.

The terms "immunodominant epitope" (claims 1, 5, 6, 13, 14, 18 and 19) and "neutralization epitope" (claim 6) are indefinite. The specification defines "immunodominant epitope" as "a portion of a protein which may be a linear arrangement of amino acids, or may be a non-linear three-dimensional arrangement of amino acids in a protein which is sufficiently exposed as to induce antibodies thereto. Such an epitope may be a 'neutralization' or 'fusion epitope' to which antibodies which bind and block infection and/or block fusion of cells" (page 22, lines 7-11). The term "immunodominant epitope" is used in the art to mean an epitope of a protein is able to induce a greater cellular or humoral immune response as compared to other epitopes. The terms "neutralization epitope" or "fusion epitope" are not defined in the specification and do not have art accepted meaning. Therefore, it is unclear whether applicants are attempting to redefine the term "immunodominant epitope" limiting the term to epitopes that just induce an antibody response or to epitopes with one particular function or structure. The metes and bounds of which epitopes applicants consider "immunodominant" or "neutralization" in the instant invention cannot be determined.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

5. Claims 1-13 are rejected under 35 U.S.C. 102(b) as being anticipated by Chada (Chada et al., June 1993, J. Virology, Vol. 67, pages 3409-3417).

Chada teaches all the limitations of the methods claimed. Chada does not enable obtaining a cellular immune response that is therapeutic or prophylactic which is the only disclosed use in the instant specification for inducing a cellular immune response in a patient. The following art rejection is based on the fact that the claims do not require a vaccine, a therapeutic effect or a prophylactic effect. Therefore, the art rejection does not conflict with the enablement rejection above and is considered proper.

Chada teaches transforming fibroblasts with a vector comprising a vaccinia viral vector encoding the HIV-IIIB envelope glycoprotein with a deletion of the V3 loop and administering the cells to mice. An envelope-specific CTL response was induced against envelope determinants (page 3410; paragraph bridging columns 1 and 2; column 2, first full paragraph; column 1, line 1; page 3411, column 1, second paragraph; paragraph bridging 3411 and 3412). Deletion of the V3 loop is considered a "modified" epitope. The deletion of the V3 loop taught by Chada appears to be equivalent to the deletion performed in the specification (see 112/1st rejection). The V3 loop is also considered a "neutralization epitope" (claim 6; see also the 112/2nd regarding "neutralization epitope").

The envelope-specific CTL response is considered a cellular immune response against a "conserved epitope" as claimed because the envelope protein is found in a number of viruses. Without evidence to the contrary, the method of Chada inherently results in cellular immunity "to a conserved epitope" of the glycoprotein (claim 1), stimulation of PBMCs (claim 7), increased CTL activity against conserved epitopes... (claim 8) and cellular immunity without inducing apoptosis of CD4+ T cells (claim 12) because the modification of the V3 loop taught by Chada is equivalent to the modification in the specification, because Chada teaches a cellular immune response against the envelope determinants, and because the transduced fibroblast are functionally equivalent to the cells of the instant invention.

Fibroblasts present envelope glycoproteins to the immune system; therefore, they are considered APC as claimed. Without evidence to the contrary, the transduced fibroblast of Chada are inherently resistant to ADCC (claim 9), do not form syncytia (claim 10) and do not undergo apoptosis (claim 11) because the cells are functionally equivalent to the cells disclosed in the specification; they express the same protein to the same cells using the same MHC molecule. Thus, Chada anticipates all the limitations of the claims.

Claims 14-18 appear to be free of the prior art of record because the prior art of record did not enable combining a nucleic acid encoding an envelope glycoprotein as claimed with an adjuvant such that a vaccine is made. Claims 19 and 20 appear to be free of the prior art of

record because the prior art of record did not teach or suggest combining cells expressing an envelope glycoprotein containing a modified immunodominant epitope with an adjuvant.

No claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson whose telephone number is (703) 305-0120. The examiner can normally be reached on Monday through Friday from 8:30 am to 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, John LeGuyader, can be reached on (703) 308-0447. The fax phone number for this Group is (703) 308-8724.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 305-0196.

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